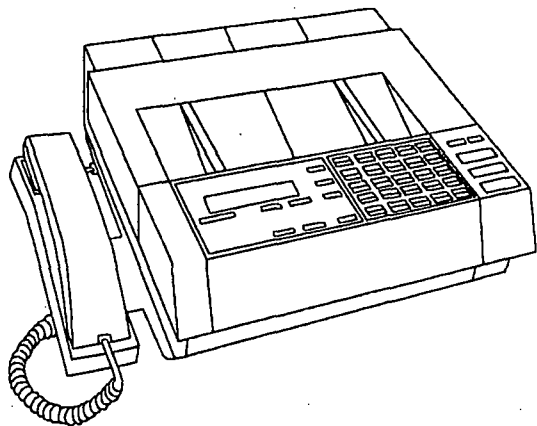


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FROM: Joan Fisk, 703/603-8791 (tel)NUMBER OF PAGES (INCLUDING COVER PAGE): 9COMMENTS: *Will be poor copy - sorry
I know Jean Dewert, one of the authors. She is truly
an expert in this area.*FROM Fax number
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3. Environmental Radiological Dose Assessment

authors:

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A. Overview of Programs

Radiological dose equivalents show the potential doses received by individuals exposed to radioactivity in the environment. Dose equivalent refers to the quantity of radiation energy absorbed per unit mass (the dose), multiplied by adjustment factors for the type of radiation absorbed. The effective dose equivalent (EDE), or dose, is the principal measurement used in radiation protection. The EDE is a hypothetical whole-body dose equivalent that would equal the same risk of cancer mortality and serious genetic disorder as the sum of the weighted dose equivalents of those organs considered to be most seriously affected by the radionuclide in question. The EDE includes the committed effective dose equivalent (CEDE) from internal deposition of radionuclides and the EDE due to penetrating radiation from sources external to the body.

Federal government standards limit the EDE to the public (DOE Order 5400.5, 40 Code of Federal Regulations [CFR] Part 61) (DOE 1990). The Department of Energy's (DOE's) public dose limit (PDL) is 100 mrem/yr EDE received from all pathways (i.e., ways in which people can be exposed to radiation, such as inhalation, ingestion, and immersion in water or air containing radioactive materials), and the dose received through the air pathway is restricted by the Environmental Protection Agency's (EPA's) effective dose standard of 10 mrem/yr (see Appendix A). These values are in addition to exposures from normal background, consumer products, and medical sources. The standards apply to locations of maximum probable exposure to an individual in an off-site, uncontrolled area.

B. Radiological Dose Equivalents

1. Methods for Dose Calculation

a. **Introduction.** Annual radiation doses are evaluated for three principal exposure pathways: external exposure (which includes exposure from immersion in air containing photon-emitting radionuclides and direct and scattered penetrating radiation), inhalation, and ingestion.

Two evaluations of potential releases are conducted: one to satisfy 40 CFR Part 61 requirements and one for all pathways. Results of environmental measurements are used as much as possible in assessing doses to individual members of the public. Calculations based on these measurements follow procedures recommended by federal agencies to determine radiation doses (DOE 1991, NRC 1977). If the impact of Laboratory operations is not detected by environmental measurements, individual and population doses attributable to Laboratory activities are estimated through computer modeling of releases.

The dose conversion factors used for inhalation and ingestion calculations are those recommended by the DOE (1988) and are based on factors in Publication 30 of the International Commission on Radiological Protection (ICRP 1979). Dose conversion factors for inhalation assume a particle size of 1- μ m-activity median aerodynamic diameter as well as the lung solubility category that will maximize the EDE (for comparison with DOE's 100 mrem/yr PDL). Similarly, the ingestion dose conversion factors are chosen to maximize the EDE for comparison with DOE's 100 mrem/yr PDL for all pathways. These dose conversion factors give the 50-year dose commitment for internal exposure. The 50-year dose commitment is the total dose received by an organ during the 50-year period following the intake of a radionuclide.

External doses from ambient air concentrations are calculated using the dose-rate conversion factors published by DOE (1988). These factors give the photon dose rate in millirem (mrem) per year per unit radionuclide air concentration in microcuries per cubic meter (μ Ci/m³). If the conversion factor for a specific radionuclide of interest is not published in DOE 1988, it is calculated with the computer program DOSFACTOR II (Kocher 1981).

b. **External Radiation.** The Laboratory's largest contributor to the penetrating radiation environment is the Los Alamos Neutron Science Center (LANSCE), formerly called the Los Alamos Meson Physics Facility. During experimentation at LANSCE, short-lived positron emitters are released from the stacks and diffuse from the buildings. These emitters release photon radiation as they decay, producing a potential external radiation dose. Most of the emitters decay very quickly, and within a few hundred meters the dose is negligible. However, the dose at East Gate (the Laboratory boundary north-northeast of LANSCE) is elevated by these Laboratory emissions. The Laboratory's contribution to the penetrating radiation dose at East Gate is derived in two ways: in

3. Environmental Radiological Dose Assessment

one method, data from a high-pressure ion chamber are used to develop a direct evaluation of the penetrating radiation exposure rate; in the other method, calculated or measured emissions from the stacks and buildings at LANSCE are input to CAP-88 to model the potential dose at East Gate. The modeling is conservative and generally results in an overestimation of the Laboratory's contribution to the hypothetical maximally exposed individual (MEI) at East Gate. Other locations in the townsite are also modeled to determine potential doses from LANSCE operations.

The other potentially significant contributor to penetrating radiation exposures is the Criticality Facility at TA-18. Criticality experiments produce neutrons and photons, both of which contribute to the external penetrating radiation dose. During experiments that have the potential to produce a dose in excess of 1 mrem per operation, public access is restricted by closing Pajarito Road from White Rock to TA-51.

Environmental thermoluminescent dosimeters (TLD) are used to estimate external penetrating radiation doses. The Laboratory has a network of TLDs (TLDNET) around the Laboratory and townsite. The large variations in the natural background levels of penetrating radiation limit the ability of TLDs to discern the low-level Laboratory releases from natural background fluctuations. However, in the event of releases of penetrating radiation significantly above background, TLDs may be used as an indicator of the magnitude of the exposures. TLDs near the TA-18 facility have shown exposure levels above background as discussed further in Section 4.B.3. The Laboratory's TLDNET is not sensitive enough to reliably distinguish LANSCE emissions from background.

The TLDNET data are used to quantify the exposure from penetrating radiation in the Los Alamos area. The modeled dose contribution from LANSCE is subtracted from the measured TLD exposures to derive the net, nonradon, background dose at a number of locations in the Los Alamos area. The final, individual, nonradon dose is derived by reducing the measured exposure by 20% to account for building shielding and by 30% to account for the self-shielding of the body. The dose from self-irradiation, caused by natural radioactive emitters such as potassium-40 within the body, is about 40 mrem annually and is also factored into the calculation. (Note: these reductions are not used for demonstrating compliance with the EPA standard.) An assumed dose of 200 mrem to account for radon exposure is added to the calculated net dose to determine the total average background dose to a person residing in the Los Alamos area.

c. **Inhalation Dose Equivalent.** Annual average air concentrations of tritium; plutonium-238; plutonium-239,240; uranium-234; uranium-235; uranium-238; and americium-241, determined by the Laboratory's air monitoring network (AIRNET), are corrected for background by subtracting the average concentrations measured at representative background stations. The net concentration is reduced by 10% to account for indoor occupancy (Kocher 1980). These net concentrations are then multiplied by a standard breathing rate of 8,400 m³/yr (ICRP 1975) to determine total adjusted intake by inhalation, in microcuries per year, for each radionuclide. Each intake is multiplied by appropriate dose conversion factors to convert radionuclide intake into 50-year committed dose equivalents (CDE). Following ICRP methods, doses are calculated for each organ that contributes more than 10% of the total EDE for each radionuclide. The dose calculated for inhalation of tritium is approximately one-half of the total dose received by being in an environment with tritium; the other half comes from direct absorption of tritium through the skin. The dose conversion factors (DCFs) for inhalation of tritium incorporate the dose received by absorption through the skin.

This procedure for dose calculation assumes conservatively that a hypothetical individual is exposed to the measured air concentration continuously throughout the entire year (8,760 h). This assumption is made for the boundary dose, dose to the MEI, and dose to the population living within 80 km (50 mi) of the site.

d. **Ingestion Dose.** Radioanalytical data from samples of foodstuffs are used to estimate the annual CDE to various tissues in the body and the total CEDE to the whole body for the average and maximum consumer of food products within the general population. The EPA's model CAP-88 also provides an estimate of the CEDE to the whole body for the air pathway only. The estimated CEDE is included in the total modeled EDE reported in Section 3.B.3.b. However, the CEDE from food products is calculated by multiplying the CDE, representing the total dose which an organ or tissue of the body is expected to receive over the 50-year period following an intake of radioactive material, by the weighting factors for that tissue as given in ICRP 26 (ICRP 1977). The CDE (and thus the CEDE) does not include contributions from exposures external to the body.

To calculate the CEDE, the radionuclide concentration in a particular foodstuff is multiplied by an estimated annual consumption rate to obtain the total adjusted intake for a particular radionuclide. The estimated annual consumption rates used for these calculations are presented in Table 3-1. Multiplication of this annual adjusted

3. Environmental Radiological Dose Assessment

intake by the appropriate radionuclide dose conversion factor for a particular organ gives the estimated CDE to the organ and, similarly the CEDE to the entire body [DOE 1988]. To determine the Laboratory impacts, if any, on a particular foodstuff, the maximum CEDE (i.e., average CEDE + two sigma) at regional stations or other background stations is subtracted from the maximum CEDE at each monitoring location. Since one cannot have a "negative exposure to radiation," all negative values are set to zero leaving only the net positive differences between the sampling location of interest and the background stations. This net positive difference is summed over all the monitored radionuclides to obtain the total net positive difference which is expressed in mrem. The total net positive difference is also reported as a percentage of the DOE's 100 mrem/yr PDL (DOE 1990) and is used to calculate the risk of cancer fatalities from consuming a particular foodstuff.

2. Estimation of Radiation Dose Equivalents

a. **Dose Equivalents from Natural Background.** Published EDE values from natural background and from medical and dental uses of radiation are used to provide a comparison with doses resulting from Laboratory operations. Global fallout doses due to atmospheric testing of nuclear weapons are only a small fraction of total background doses (<0.3% [NCRP 1987a]). Natural background radiation dose is due to exposure to the lungs from radon decay products and exposures from nonradon sources which affect the whole body.

External radiation comes from two sources of approximately equal magnitude: the cosmic radiation from space and terrestrial gamma radiation from radionuclides in the environment. Estimates of background radiation are based on a comprehensive report by the National Council on Radiation Protection and Measurements (NCRP 1987b). The 1987 NCRP report uses 20% shielding by structures for high-energy cosmic radiation and 30% self-shielding by the body for terrestrial radiation. The 30% protection factor is also applied to less energetic gamma radiation from LANL sources.

Whole-body external dose is incurred from exposure to cosmic rays, external terrestrial radiation from naturally occurring radioactivity in the earth's surface, and from global fallout. The EDE from internal radiation is due to radionuclides naturally present in the body and inhaled and ingested radionuclides of natural origin.

Annual external background radiation exposures for sources other than radon vary depending on factors such as snow cover and the solar cycle (NCRP 1975b). Estimates of background radiation in 1995 from nonradon sources are based on environmental dosimeter measurements of 109 mrem in Los Alamos and 96 mrem in White Rock using only complete datasets (i.e., measurements for all four quarters). The elevation difference between Los Alamos and White Rock accounts mainly for the difference between the two numbers. These measured doses were adjusted for structural shielding by reducing the cosmic ray component by 20%. The measured doses were also adjusted for self-shielding by the body by reducing the terrestrial component by 30%. The neutron dose from cosmic radiation and the dose from self-irradiation were then included to obtain the whole-body background dose of 149 mrem at Los Alamos and 136 mrem at White Rock from sources other than radon. Uranium decay products occur naturally in soil and building construction materials. Inhalation of radon-222 produced by decay of radium-226, a member of the uranium series, results in a dose to the lung, which also must be considered. The EDE from radon-222 decay products is assumed to be equal to the national average, 200 mrem/yr. This estimate may be revised if a nationwide study of background levels of radon-222 in homes is undertaken. Such a national survey has been recommended by the NCRP (NCRP 1984, 1987a).

In 1995 the EDE to residents was 349 mrem at Los Alamos and 336 mrem at White Rock from all natural sources. The individual components of the background dose for Los Alamos and White Rock, and the average EDE of 53 mrem/yr to members of the US population from medical and dental uses of radiation (NCRP 1987a) are listed in Table 3-2.

b. Summary of Doses to the Public from Laboratory Operations

Inhalation of Airborne Emissions. The net CEDE from the inhalation of airborne emissions as measured by the AIRNET in 1995 for the townsites of Los Alamos and White Rock are 0.05 mrem and 0.06 mrem, respectively. The maximum potential CEDE from TA-54, Area G operations, from explosive testing containing depleted uranium, and from decontamination and decommissioning activities at TA-21 are estimated at 0.002 mrem, 0.04 mrem, and 0.006 mrem, respectively. These potential doses to the public are well below the EPA standard of 10 mrem/yr for airborne emissions [EPA 1989]. Section 4.B.1.c provides further discussions on the CEDE by sampling locations as well as the radionuclides that contributed to this dose estimate.

3. Environmental Radiological Dose Assessment

External Penetrating Radiation from Airborne Emissions and Direct Sources. The annual EDE for airborne emissions was measured near the location of the MEI along the LANL boundary known as East Gate. The above background EDE at this location in 1995 was 2.0 mrem. No direct penetrating radiation dose to the public from Laboratory operations was detected by TLD measurements. Section 4.B.3.e provides further discussions on the EDE by sampling locations.

Ingestion of Drinking Water. The maximum annual CEDE (i.e., the total CEDE plus two sigma for the maximum consumption rate) for drinking water samples collected in 1995 from the LANL water distribution system is 0.579 mrem (14.5% of the 4-mrem drinking water standard). The maximum annual CEDE for the average consumption rate decreases to 0.411 mrem (10.3% of the 4-mrem drinking water standard). Section 5.C.4 provides further discussions on the CEDE for Los Alamos and White Rock and the Pueblos of San Ildefonso, Santa Clara, Cochiti, and Jemez.

Exposure to Sediments in Mortandad Canyon. The pathways of exposure evaluated for sediment sampling in Mortandad Canyon include the external gamma pathway from radioactive material deposited in the sediments, the inhalation pathway from materials resuspended by winds, animals, etc.; and the soil ingestion pathway. Using RESRAD v 5.61, the maximum total effective dose equivalent (TEDE) (i.e., the total of the EDEs from all pathways plus twice the error term) is estimated as 36.6 mrem (<37% of the DOE PDL). Cesium-137 from sampling locations GS-1 and MCO-5 contributed to more than 98% of the external gamma pathway which, in turn, contributed more than 84% to the maximum TEDE for the entire canyon system. The inhalation and soil ingestion pathway each contributed approximately 8% to this maximum TEDE. Modeling assumptions and more detail information is found in Section 5.E.6.

Exposure to TA-50 Effluent and Stream Below Outfall. The maximum annual CEDE (i.e., the total CEDE plus two sigma using the maximum consumption rate of 16.1 L/yr) for water samples collected in 1995 directly from the TA-50 effluent and from the stream below the outfall is 20.9 mrem (21% of the DOE PDL) and 7.8 mrem (7.8% of the DOE PDL), respectively. For the average consumption rate of 5.7 L/yr, the annual CEDE decreases to 7.4 mrem and 2.8 mrem, respectively. Section 5.E.7 provides further discussions on the assumptions used in this calculation.

Ingestion of Foodstuffs. Using the maximum consumption rate (see Table 3-1), the maximum difference between the total positive CEDE at all sampling locations and the regional background locations for each food group is as follows: produce, 0.228 mrem; honey, 0.010 mrem; eggs, 0.002 mrem; milk, 0.063 mrem; fish (bottom feeders), 0.027 mrem; fish (higher level feeders), 0.003 mrem; elk muscle, 0.027 mrem; and elk bone, 0.216 mrem. Assuming one individual consumed the total quantity for each food group (except elk bone), the total net positive difference for the CEDE is 0.360 mrem (<0.4 % of the DOE PDL) using the maximum consumption rate and 0.081 mrem (<0.09% of the DOE PDL) using the average consumption rate.

The single factor Analysis of Variance (ANOVA) test shows that, at the 95% level of confidence, there is no significant difference between the maximum CEDE (i.e., average CEDE + two sigma) for consuming food products collected at on-site, perimeter, or off-site locations in 1995. For foodstuffs that had more than one sample per year, the Student's t Test also shows that there is no significant difference, at the 95% level of confidence, between the CEDE for 1995 and the CEDE for 1994 (or a previous collection period). For foodstuffs that had only one sample per year, the confidence interval for each dataset overlapped, also indicating there is no difference between the CEDEs for 1994 and 1995. Section 6.B.2 provides further discussions on the CEDE by the food type and sampling locations as well as the radionuclides that contributed to this total net positive difference.

3. Total Maximum Individual Dose to a Member of the Public from 1995 Laboratory Operations

a. Measured Maximum Individual Dose. The maximum individual EDE to a member of the public from 1995 Laboratory operations is estimated to be 2.3 mrem. This is the total EDE from all potential pathways of radiation exposure and is based entirely on environmental measurements. This dose is 2.3% of the DOE's annual public dose limit of 100 mrem EDE from all pathways and 1% of the total annual dose contribution from all sources of radiation (Figure 3-1). The maximum individual dose occurred at East Gate and was primarily due to exposure to external penetrating radiation from air activation products released by the LANSCE accelerator. The contribution to the maximum individual off-site dose via each pathway is presented in Figure 3-2.

b. Modeled Maximum Individual Dose. As required by the EPA, compliance with regulation 40 CFR 61, Subpart H must be demonstrated with the CAP-88 version of the computer codes PREPAR2, AIRDOS2, DARTAB2, and RADRISK (EPA 1990). These codes use measured radionuclide release rates and meteorological

3. Environmental Radiological Dose Assessment

information to calculate airborne concentrations of radionuclides released to the atmosphere. The programs estimate radiation exposures from inhalation of radioactive materials; external exposure to the radionuclides present in the atmosphere and deposited on the ground; and ingestion of radionuclides in drinking water, produce, meat, and dairy products. The source term, the amount of a particular matter, for these calculations was based on measured emissions during 1995. Wind speed, wind direction, and stability class are continuously measured at meteorology towers located at TA-54, TA-49, TA-6, and TA-53. Emissions were modeled with the wind information most representative of the release point. The maximum individual EDE from 1995 airborne emissions, as determined by CAP-88, was 5.05 mrem. The maximum dose, which would occur in the area just north-northeast of LANSCE, is 50.5% of the EPA's air pathway standard of 10 mrem/yr EDE.

c. Comparison of Department of Energy and Environmental Protection Agency Dose Methodologies.

The effects of increased dispersion of LANL's radioactive air effluents caused by the rugged topography of the Pajarito Plateau are not well incorporated by EPA's atmospheric dispersion model CAP-88. As such, the measured exposure rate at East Gate is typically less than the predicted exposure rate using CAP-88 (Figure 3-3). This is just one example of the many differences which contribute to the contrast between the dose measured for compliance to DOE standards and the dose modeled for compliance to EPA regulations presented above:

4. Population Distribution

The population distribution is used to calculate the collective dose resulting from 1995 Laboratory operations. In 1995, the estimated population of Los Alamos County was approximately 18,000 (BBER 1995). Two residential and a few commercial areas exist in the county (Figure 1-1). The Los Alamos townsite (the original area of development) now includes residential areas known as Eastern Area, Western Area, North Community, Barranca Mesa, and North Mesa. The townsite had an estimated population of 12,000 residents. The White Rock area includes the residential areas of White Rock, La Senda, and Pajarito Acres. The area had about 6,000 residents in mid-1995. It is estimated that over 241,000 persons lived within an 80-km (50-mi) radius of the Laboratory in mid-1995 (Table 3-3).

5. Collective Dose

The collective EDE from 1995 Laboratory operations is the sum of the estimated dose received by each member of the population within an 80-km (50-mi) radius of LANL. Over 99% of this dose is expected to have resulted from airborne radioactive emissions from Laboratory programs. As a result, the collective dose was estimated by modeling 1995 radioactive air emissions, their transport off site, and the resulting radiation exposures that could occur. The distribution given in Table 3-3 was used in the dose calculation. The collective dose was calculated with the CAP-88 collection of computer programs. These programs were also used to calculate the maximum EDE to a member of the public as required by the EPA regulations in 40 CFR Part 61. Airborne radioactive emissions from all types of releases were included in the analysis. The same exposure pathways that were evaluated for the maximum individual dose were also evaluated for the collective dose; these pathways include inhalation of radioactive materials, external radiation from materials present in the atmosphere and deposited on the ground, and ingestion of radionuclides in meat, produce, and dairy products. The 1995 population collective EDE attributable to Laboratory operations to persons living within 80 km (50 mi) of the Laboratory was calculated to be 3.2 person-rem. This dose is less than 0.004% of the 82,000 person-rem annual average exposure from natural background radiation and less than 0.03% of the 12,800 person-rem exposure an average person receives annually from medical radiation.

C. Risk to an Individual from Laboratory Operations

1. Estimating Risk

Health effects from radiation exposure (primarily cancer) are observed in humans only at doses in excess of 10 rem delivered at high dose rates (HPS 1996). In past environmental surveillance reports, our practice has been to use the risk estimates, also called risk factors, presented in the BEIR documents (most recently, BEIR V 1990) to quantify the cancer risks from exposure to radiation. These risks were presented to provide a perspective on the potential risk of cancer from Laboratory contributions to the radiation environment of northern New Mexico.

3. Environmental Radiological Dose Assessment

Although it is important to address the potential risk from these radiation doses, it is also important not to mislead the reader into concluding that small radiation doses are more hazardous than they actually are.

The risk estimates in BEIR V were developed by the National Academy of Sciences and were based primarily on the dose-risk effects produced in survivors of the Hiroshima and Nagasaki atomic bomb blasts. These calculations, however, overestimate actual risk for low linear energy transfer (low-LET) radiation, which is the source of more than 95% of the dose to the MBL from Laboratory operations. The NCRP (1975a) has warned that "risk estimates for radiogenic cancers at low doses and low dose rates derived on the basis of linear (proportional) extrapolation from the rising portions of the dose incidence curve at high doses and high dose rates . . . cannot be expected to provide realistic estimates of the actual risks from low-level, low-LET radiation and have such a high probability of overestimating the actual risk as to be of only marginal value, if any, for purposes of realistic risk-benefit evaluation." The fundamental shortcoming of the BEIR V risk estimates for determining low-level radiation effects is that they are based, primarily, on the effects of doses of tens or hundreds of rem received over periods of seconds. Extrapolating these data linearly downward to the mrem or fractions of mrem annual doses from Laboratory operations almost certainly results in a great overestimation of risk.

As early as the 1920's, investigators concluded that low levels of radiation could not cause the mutations and other effects assigned to such doses (Muller 1935). More recently, Billen (1990) concluded that radiation-induced DNA damage is a small contributor to the ongoing, spontaneous DNA damage that occurs in mammalian cells. In Billen's discussion, he suggests that an annual dose in the range of less than or equal to 100 mrem can be considered a "negligible dose." In terms of DNA damage, this dose is so small as to provide no effect that could be discerned from other causes. Other researchers conclude that there is no scientific basis for the low-dose risk estimates recommended by the EPA and BEIR V, and instead, propose new risk assessment methodologies that involve defining minimum significant risk (Seiler 1994 and Seiler 1996).

Radiation hormesis (the concept that small radiation doses in the range of a few rem annually may be beneficial) should also be considered when evaluating radiation-induced risk. The following discussion is paraphrased from Gollnick (1994). The descriptor *beneficial* means that a population exposed to small amounts of radiation will experience fewer cancer deaths than a similar, unexposed population. Among the claimed effects of small radiation doses, in addition to the potential for reduced cancer risk, are increased life span, growth, and fertility. Gollnick describes possible biochemical bases for these effects including elevated antibody levels in irradiated animals and differential sensitivity of different types of lymphocytes to radiation which effectively increase the body's ability to attack tumors. Some population studies support the radiation hormesis concept, although there are generally too many potential conflicting or contributing factors to draw indisputable conclusions.

Recently, the Health Physics Society (HPS) published a position statement on the risks of radiation exposures (HPS 1996). They recommended "against quantitative estimation of health risk below an individual dose of 5 rem in one year. . ." They concluded that below an individual dose of 5 rem in one year "risk estimates should not be used; expressions of risk should only be qualitative emphasizing the inability to detect any increased health detriment (i.e., zero health effects is the most likely outcome)."

Risk estimates range from 5×10^{-7} excess cancer deaths per mrem to members of the public (EPA 1994) to a negative (beneficial), although unquantified risk. We present the range of risk estimates in this section to allow readers to draw their own conclusions regarding the dangers of Laboratory radiation. If one chooses to use the BEIR or EPA risk estimates (factors) to calculate the potential excess cancer rates from a radiation dose, the result will overestimate the actual risk. The potential excess cancer deaths may be calculated according to the following equation:

$$R = D \times RF$$

where

R = incremental (or decremental) risk of cancer death expected from a radiation dose to an individual,

D = effective dose equivalent (mrem), and

RF = risk factor (excess cancer deaths/mrem).

As noted previously, RFs range from 5×10^{-7} /mrem to negative, as yet unquantified values. In the following sections, we do not report the potential risks associated with the reported doses, but the reader may calculate these according to the above equation, using whichever risk factors he/she believes to be appropriate.

3. Environmental Radiological Dose Assessment

2. Risk from Whole-Body Radiation

Radiation exposures considered in this report are of two types: (1) whole-body exposures, and (2) individual organ exposures. The primary doses from nonradon natural background radiation and from Laboratory operations are whole-body exposures. With the exception of natural background radon exposures, discussed below, radiation doses and associated risks from those radionuclides that affect only selected body organs are a small fraction of the dose and are negligible. Risks from whole-body radiation can be estimated using the factors of the BEIR V report.

Risk factors from the BEIR estimate (BEIR V 1990) are based on the risk from a single, instantaneous, high-dose-rate exposure of 10 rem. The BEIR V report states that this estimate should be reduced for an exposure distributed over time that would occur at a substantially lower dose rate. The National Academy of Sciences committee discussed dose rate effectiveness factors (DREFs) ranging from 2 to 10 that should be applied to the nonleukemia part of the risk estimate. Using the DREF value of 2 the total risk estimate from BEIR V is 440 cancer (nonleukemia and leukemia) fatalities per 10^7 person-mrem. The EPA recently recommended using a risk factor of 5×10^{-7} per person-mrem (EPA 1994) for estimating risks from whole-body radiation.

3. Risk from Exposure to Radon

Radon and radon-decay products are the largest contributors to natural background radiation exposures. These exposures differ from the whole-body radiation discussed above in that they principally involve only the localized exposure of the lung and not other organs in any significant way. Consequently, the risks from radon exposure are calculated separately. Exposure rates to radon (principally radon-222) and radon-decay products are usually measured with a special unit, the working level (WL); 1 WL corresponds to a liter of air containing short-lived radon decay products that have a total potential alpha energy of 1.3×10^5 MeV. An atmosphere having a 100 pCi/L concentration of radon-222 at equilibrium with its decay products corresponds to 1 WL. Cumulative exposure is measured in working level months (WLMs). A WLM is equal to exposure to 1 WL for 170 hours.

The estimated national-average radon EDE that was given by the NCRP is 200 mrem/yr. The NCRP derived this dose from an estimated national-average radon exposure of 0.2 WLM/yr. Because the risk factors are derived in terms of WLM, for the purposes of risk calculation it is more convenient to use the radon exposure of 0.2 WLM/yr than to use the radon dose of 200 mrem/yr. However, the 0.2 WLM/yr and the 200 mrem/yr EDE correspond to the same radiation exposure. Increased risks of fatal cancer from radon exposure can be estimated using a risk factor of 3.50×10^{-4} /WLM (BEIR IV 1988). Alternatively, on the basis of other data (Gollnick 1994), one may assume a zero or negative risk factor for exposure to radon.

4. Risk from Nonradon Natural Background Radiation

During 1995, persons living in Los Alamos and White Rock received an average EDE of 149 mrem and 136 mrem, respectively, of nonradon radiation (principally to the whole body) from natural sources (including cosmic, terrestrial, and self-irradiation sources, with allowances for shielding and cosmic neutron exposure) (Table 3-2).

The dose from natural background radiation also includes exposure to the lung from radon-222 and its decay products as discussed above.

5. Risk from Laboratory Operations

The risks calculated from natural background radiation and medical and dental radiation can be compared with the incremental risk caused by radiation from Laboratory operations. The average doses to individuals in Los Alamos and White Rock from 1995 Laboratory activities were 0.5 and 0.2 mrem, respectively. Assuming the EPA risk factors, these Laboratory doses would give approximately 0.1% of the risk attributed to exposure to natural background radiation or to medical and dental radiation. The exposure to Los Alamos County residents from Laboratory operations is well within variations in exposure of these people to natural cosmic and terrestrial sources and global fallout. For example, variation in the amount of snow cover and in the solar sunspot cycle can cause a 10-mrem difference from year to year (NCRP 1975b).

For Americans, the average lifetime risk is a 1-in-4 chance of contracting cancer and a 1-in-5 chance of dying of cancer (EPA 1979). Assuming one accepts the most conservative risk estimates (BEIR V 1990 and EPA 1994), the incremental risk from exposure to Laboratory operations is negligible.



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COMMENTS:

This is very interesting! When I was in Los Alamos I had all this stuff, but did not really think much about it - except I shouldn't worry! It will be loosey copy (a fax of a fax I got from a friend in Los Alamos), but gives quite a message. I marked a few areas!

Pages 70 & 71 are especially interesting. I will be e-mailing you some stuff also.

John